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CLINICAL USE OF CARDIAC STRESS PERFUSION MRI SCAN TO GUIDE TREATMENT OF NON CULPRIT CORONARY ARTERY DISEASE IN PATIENTS WITH MULTI-VESSEL DISEASE UNDERGOING PPCI FOR STEMI

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Background The primary objective of primary percutaneous coronary intervention (PPCI) in patients with ST-segment elevation myocardial infarction (STEMI) is to restore epicardial flow and myocardial perfusion in the culprit vessel. It is estimated that up to 65% of the patients presenting with STEMI have multi-vessel

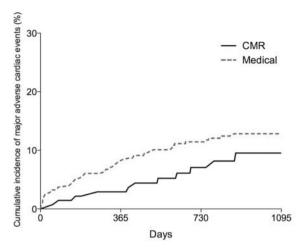


Figure 1

disease (MVD), currently it remains uncertain whether treatment of non-culprit vessels is required and when this should be done with on-going trials exploring this question. With the development of adenosine stress CMR it has become possible to assess the perfusion of the myocardium in more detail and offer an alternative diagnostic strategy for non-culprit disease. The aim of this study was to look at the use of CMR to guide intervention to non-culprit disease in patients with MVD undergoing PPCI.

Methods This was an observational cohort study of 983 patients with multi-vessel disease who underwent PPCI from 2007 to 2011. Patients with previous CABG, cardiogenic shock and those undergoing complete or staged revascularisation were excluded. The remaining 560 patients were split into 2 groups, 430 patients who were managed medically and 130 patients who had Cardiac MRI perfusion scan to guide further revascularisation in addition to standard medical therapy. The primary outcome was major adverse cardiac events (all cause mortality, myocardial infarction, target vessel revascularisation and stroke). Follow-up was for a median of 2.7 years (IQR range 1.8–4.1 years).

Results From 130 patients who underwent stress CMR, 82 (63%) patients had perfusion defects. 18 (13%) of which were matched perfusion defects (with infarct area) and 64 (49%) had unmatched perfusion defects. From the patients who had unmatched perfusion defects 24 (18.5%) went on to have further PCI to non-culprit vessels, 4 (3%) underwent planned CABG, with the remaining 36 (27.7%) managed medically. Patients with both matched and no perfusion defects were also managed medically. Patients who underwent a CMR were younger than those treated medically (57.5 vs 66.0. p<0.0001), otherwise there were similar baseline characteristics between the groups. Over the follow-up period there was a trend to lower MACE rates in the CMR group driven by lower rates of unscheduled revascularisation and myocardial infarction however this was not statistically significant (p=0.205). Univariate Cox analysis revealed no association between CMR imaging and MACE rates (HR 0.70 (95% CI 0.38 to 1.31), however after multivariate adjustment (HR 0.46 (95% CI 0.22 to 0.97), CMR guided management did appear to reduce 3 year MACE events.

Conclusions MACE rates in patients who had stress CMR to guide treatment of non-culprit disease were lower compared to medical management. It was noted that patients who were treated medically without a CMR scan were older with possible other co-morbidities. Further randomised study is needed to explore the role of stress CMR scanning in guiding treatment of non culprit coronary artery disease in patients with multi vessel disease.

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